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EXPERIMENTAL STUDIES

Definition of the Safe Lower Limits of Aortic Resection During Surgical Procedures on the Thoracoabdominal Aorta: Use of Somatosensory Evoked Potentials

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The technique of intraoperative monitoring of somatosensory evoked potentials was applied to a canine model of spinal cord ischemia in an attempt to determine the safe lower limits of aortic resection during thoracic aortic surgery. Fifteen animals underwent left thoracotomy with institution of partial left atrial/femoral artery bypass for maintenance of distal aortic perfusion after proximal descending thoracic aortic exclusion. In Group I animals (n = 6, control), no further interventions were performed so that the effect of exclusion of vessels noncritical to spinal cord blood supply could be assessed by measurements of spinal cord blood flow and somatosensory evoked potentials. In Group II animals (n = 8), the level of distal aortic exclusion was progressively lowered until loss of somatosensory evoked potential (critical vessel exclusion) occurred. The effect of critical vessel exclusion on spinal cord blood flow was then assessed.

Exclusion of multiple vessels noncritical to spinal cord blood supply (Group I) had no effect on spinal cord blood flow or function (somatosensory evoked potentials). Exclusion of vessels critical to spinal cord blood supply resulted in significant spinal cord ischemia (83.4% flow reduction, probability [p] < 0.05 versus baseline) and ischemic spinal cord dysfunction (loss of somatosensory evoked potential). The data indicate that monitoring of intraoperative somatosensory evoked potentials allows recognition of spinal cord ischemia due to exclusion of vessels critical to spinal cord blood supply and, thus, is an accurate predictor of the safe lower limits of aortic resection performed during surgical procedures on the descending thoracic or the thoracoabdominal aorta, or both.

The necessity for routine ligation and exclusion of multiple intercostal and lumbar vessels during the course of operative repair of lesions of the descending thoracic and thoracoabdominal aorta may result in the permanent, though inadvertent, interruption of vessels critical to the blood supply of the spinal cord and subsequent paraplegia. Such catastrophic neurologic complications have been, up to now, unpreventable. The anatomic blood supply to the spinal cord is highly variable. Preoperative angiographic identification of these critical vessels is rare, and to date, there has been no reliable method for intraoperative localization of these arteries (1–5).

Numerous adjuncts (temporary shunt and bypass techniques) have been introduced in attempts to improve distal organ perfusion and prevent paraplegia after proximal aortic cross-clamping (6–11). However, these devices are ineffectual in preventing spinal cord injury if, during the course of the operative repair, vessels supplying the cord are excluded and permanently interrupted. Although clinical evidence suggests that preservation or reimplantation, or both, of intercostal and lumbar vessels may decrease the incidence of paraplegia (1,11), such procedures are not reliable as a result of the present inability to accurately localize vessels critical to spinal cord blood supply. Therefore, we employed the technique of intraoperative somatosensory evoked potential monitoring in order to determine if this method could be used to define the safe lower limits of aortic resection by allowing recognition of spinal cord ischemia due to the exclusion of vessels critical to spinal cord blood supply.

Methods

Experimental preparation. Fifteen dogs weighing 25 to 30 kg were anesthetized with morphine sulfate (2.5 mg/kg body weight) and alpha chloralose (100 mg/kg). After intubation, respiration was controlled with room air positive

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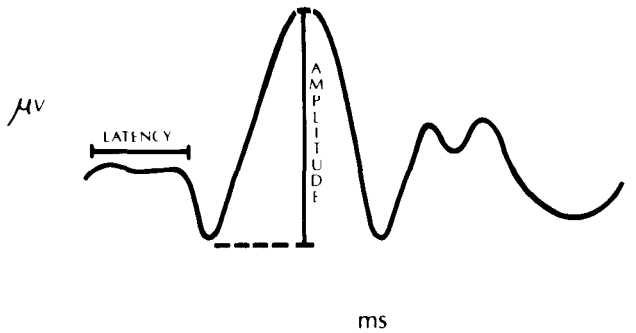
pressure ventilation and low flow oxygen. A left thoracotomy was performed and the heart suspended in a pericardial cradle. Catheters placed in the aortic root were used for pressure measurements, serial blood sampling and collection of reference samples for determination of spinal cord blood flow by the radioactive microsphere technique (12). Additional pressure measurement catheters were placed in the left ventricle, left atrium and femoral artery. Radioactive microspheres (iodine-125, cerium-141, strontium-85, scandium-46) were injected through the left atrial catheter.

Evoked potential studies. Somatosensory evoked potentials were measured by means of a clinical evoked potential system (TN-3000, TRACOR Analytic, Inc.) which has been used to monitor spinal cord conduction. Somatosensory evoked potential traces were generated by stimulation of the left posterior tibial nerve with a bipolar input channel. After conduction of the impulses through the dorsal spinal columns, the cortical response to 200 consecutive stimuli were recorded from needle electrodes placed in the scalp at the nasion and 55% of the distance from the nasion to the inion in the midline of the scalp. A separate grounding electrode was placed in the left ear. The potentials were amplified 10,000 × and then processed with a 10 Hz low pass and a 250 Hz high pass filter. To improve the signal to noise ratio of these small potentials, 200 consecutive responses activated by supramaximal stimuli to the nerve (4× the motor twitch threshold, 20 mA, 0.4 ms duration pulses, 2.3/s) were averaged for each trace. A separate grounding electrode was placed in the upper thorax of each animal and, in addition, a no stimulus control trace was recorded in each experiment for the establishment of background noise levels.

A typical somatosensory evoked potential trace is shown in Figure 1. Two variables were serially monitored: latency of onset and amplitude of the generated response. Increases in latency or decreases in amplitude, or both, of the somatosensory evoked potentials indicate spinal cord ischemia (13,14). Generation of new traces every 2 minutes for comparison with baseline will demonstrate changes in these variables due to ischemia. All somatosensory evoked potential data were stored in a computer for subsequent retrieval and comparison with baseline.

Figure 1. Typical somatosensory evoked potential trace.



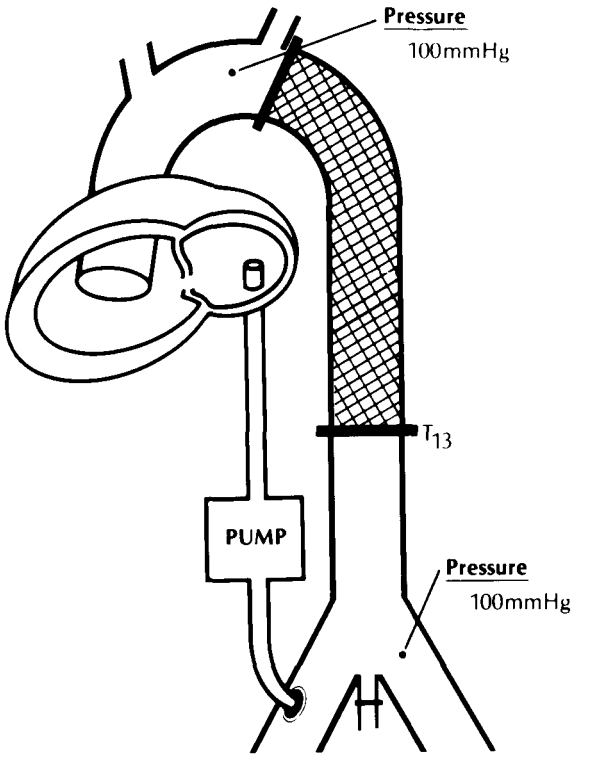


Figure 2. Graphic summary of the experimental protocol used in Group I dogs. See text for details.

Experimental protocol. After baseline determinations of spinal cord blood flow and somatosensory evoked potentials, aortic cross clamps were placed in all animals just distal to the origin of the left subclavian artery. In Group I dogs (n = 6), the distal aorta was cross-clamped at the level of T_{13} and the mid-sacral artery was ligated (Fig. 2). The distal aorta was cross-clamped in Group II dogs (n = 9), at the level of T_7 without ligation of the mid-sacral artery (Fig. 3). Partial left atrial/femoral artery bypass was then instituted in all animals for maintenance of distal aortic perfusion (mean perfusion pressure of 100 mm Hg). After a 30 minute stabilization period, spinal cord blood flow and somatosensory evoked potential determinations were repeated for comparison with baseline. Group I animals underwent no further intervention.

In Group II dogs, paired aortic cross clamps were then placed as depicted in Figure 3, so that progressively lower aortic segments were excluded at 15 minute intervals until somatosensory evoked potential loss occurred. Twenty minutes later, spinal cord blood flow measurements were repeated for comparison with baseline. Perfusion pressure in the most distal nonexcluded segment was maintained at a mean pressure of 100 mm Hg throughout the entire experiment with the partial left atrial/femoral artery bypass pump. This assured normal blood flow to any distal vessels, and

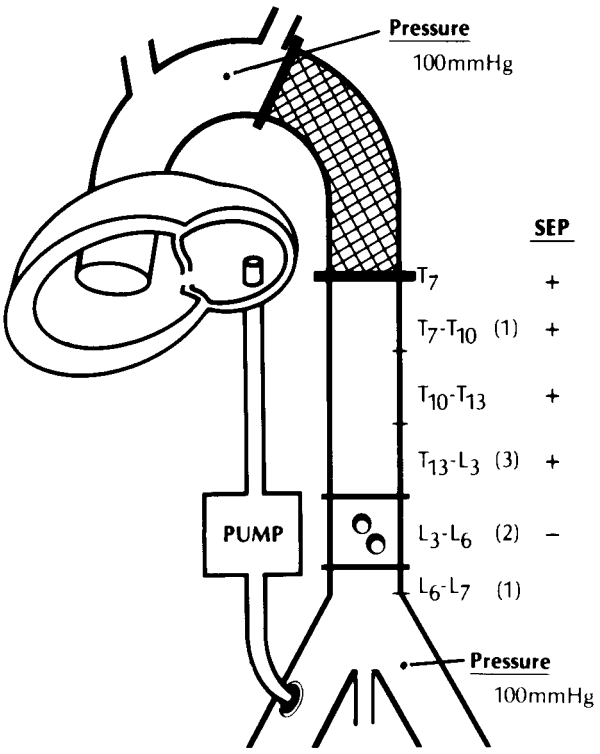


Figure 3. Graphic summary of the experimental protocol used in Group II dogs. The level at which the aorta was excluded at the time of somatosensory evoked potential loss was highly variable. Exclusion of the L_3 - L_6 aortic segment depicted here is only used as an example. The actual number of animals losing somatosensory evoked potential at each specific aortic level of exclusion is shown in parentheses. SEP = somatosensory evoked potential.

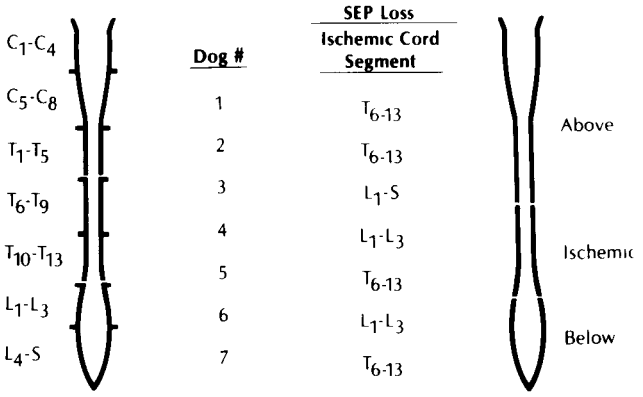


Figure 4. A method for interpretation of spinal cord blood flow studies. The spinal cord on the **left** depicts its division into 7 segments on removal. (C = cervical; T = thoracic; L = lumbar; S = sacral). For Group I animals, spinal cord blood flow changes were analyzed for the upper (C_1–T_5), mid (T_6–T_{13}), and lower (L_1–S) spinal cord regions as shown on the **left**. Interpretation of regional spinal cord blood flow studies in Group II animals (spinal cord on **right**) was based on segmental spinal cord blood flow studies obtained at the time of evoked potential loss. See text for details. SEP = somatosensory evoked potential.

prevented peripheral nerve ischemia. The mid-sacral artery was not ligated in Group II animals because important spinal radicular arteries may originate from this vessel in dogs.

In Group II animals (n = 2) in which no somatosensory evoked potential changes occurred after exclusion of the entire descending thoracic and thoracoabdominal aorta (left subclavian–L_7), two different radioactive tracers were injected simultaneously for determination of the origin of spinal cord blood flow. The initial bead was injected in the left ventricular cavity to determine the extent of the spinal cord supplied by the circulation originating proximal to the left subclavian artery. The second bead was simultaneously injected into the left atrial/femoral artery bypass pump circuit (reference samples collected from the femoral artery pressure cannula) to determine the extent of spinal cord tissue supplied by vessels originating from the mid-sacral artery and its branches.

Interpretation of spinal cord blood flow studies. All spinal cord blood flow studies were performed using the radioactive microsphere technique (12). After completion of all experiments, the entire spinal cord (C_1–sacrum) was excised after posterior laminectomy. Spinal cord tissue was then divided into 7 segments as depicted in Figure 4. In Group I animals, no ischemic segments were detected, and analysis of blood flow data was performed by comparing flows from the upper (C_1–T_5), mid (T_6–T_{13}) and lower (L_1–S) spinal cord regions with baseline. In the seven Group II animals in which somatosensory evoked potential loss occurred, blood flow studies performed at the time of such loss identified ischemic spinal cord segments in each animal (Fig. 4). Changes in spinal cord blood flow in these ischemic spinal cord segments, and changes simultaneously noted in all cord segments both above and below these ischemic segments, were then compared with baseline.

Analysis of spinal cord blood flow changes for Group II is based only on those dogs in whom somatosensory evoked potential loss occurred (seven of nine animals). In two of the Group II animals, somatosensory evoked potential loss did not occur despite exclusion of the entire descending and thoracoabdominal aorta (left subclavian–L_7). The results of spinal cord blood flow studies from these animals were analyzed separately.

Statistical analysis. Statistical significance was determined using analysis of variance; all values are reported as mean ± standard error of the mean.

Table 1. Effect of Noncritical Vessel Exclusion on Somatosensory Evoked Potentials in Group I Dogs (n = 6)

	Latency (ms)	Amplitude (μV)
Baseline	25.39 ± 2 35	6 48 ± 0.89
Noncritical vessel exclusion	25.28 ± 2 78	6.16 ± 1 02

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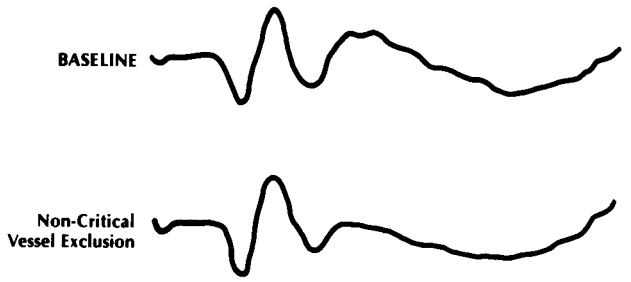

Figure 5. Graphic illustration of somatosensory evoked potential traces constructed from data in Table 1. Note the lack of any potential changes due to maintenance of spinal cord blood flow after noncritical vessel exclusion.

Results

Group I (n = 6)

Proximal exclusion (left subclavian–T_{13}) of multiple vessels noncritical to spinal cord blood flow had no effect on somatosensory evoked potentials (Table 1). Traces constructed from the data in Table 1 graphically illustrate somatosensory evoked potential preservation (Fig. 5).

The results of spinal cord blood flow studies are summarized in Table 2 and depicted in Figure 6. Proximal exclusion of multiple vessels noncritical to spinal cord blood supply had no effect on spinal cord blood flow measurements due to the maintenance of distal aortic perfusion by partial left heart bypass.

Table 2. Effect of Noncritical Vessel Exclusion on Spinal Cord Blood Flows (cc/100 g per min) in Group I Dogs (n = 6)

Spinal Cord Segments	Baseline	Noncritical Vessel Exclusion
C_1-T_5	13.28 ± 2.51	10.02 ± 1.71
T_6-T_{13}	12.71 ± 2 59	11 40 ± 0.83
L_1-S	21.89 ± 5.14	18.52 ± 3.76

C = cervical; L = lumbar; S = sacral; T = thoracic.

Group II (n = 9)

Somatosensory evoked potentials. Proximal exclusion (left subclavian–T_7) of multiple vessels noncritical to spinal cord blood flow again had no effect on somatosensory evoked potentials (Table 3). Subsequent exclusion of progressively lower aortic segments resulted in potential loss in seven of nine dogs. The level of the thoracoabdominal aorta excluded at the time of somatosensory evoked potential loss was highly variable, ranging from T_7 to L_7 (Table 4, Fig. 3). Somatosensory evoked potential loss in these animals occurred after the characteristic time-dependent decay in latency and amplitude (progressing to complete flattening) previously described with spinal cord ischemia (13,14). Traces constructed from the data in Table 3 illustrate these somatosensory evoked potential changes (Fig. 7).

Spinal cord blood flow. In each of the seven animals exhibiting somatosensory evoked potential loss, ischemic spinal cord segments were identified by radioactive microsphere studies performed at the time of evoked potential loss (Table 4, Fig. 4). The results of spinal cord blood flow studies in these animals are summarized in Table 5 and depicted in Figure 8. Proximal exclusion of multiple vessels noncritical to spinal cord blood flow (left subclavian–T_7) had no effect on blood flow due to the maintenance of distal aortic perfusion. In contrast, significant segmental ischemia ($p < 0.0125$ versus baseline) was observed in "ischemic" cord segments during critical vessel exclusion (evoked potential loss). Simultaneous determinations of spinal cord blood flow for all cord segments above and below the isolated ischemic segments showed no change from baseline throughout the experiment.

In two of nine animals, no somatosensory evoked potential changes were observed despite exclusion of the entire descending thoracic and thoracoabdominal aorta (left subclavian–L_7) (Fig. 9). In these animals, simultaneous injection of different radioactive tracers into the systemic (left ventricular injection site) and bypass (left atrial/femoral artery) circulations explained the lack of evoked potential

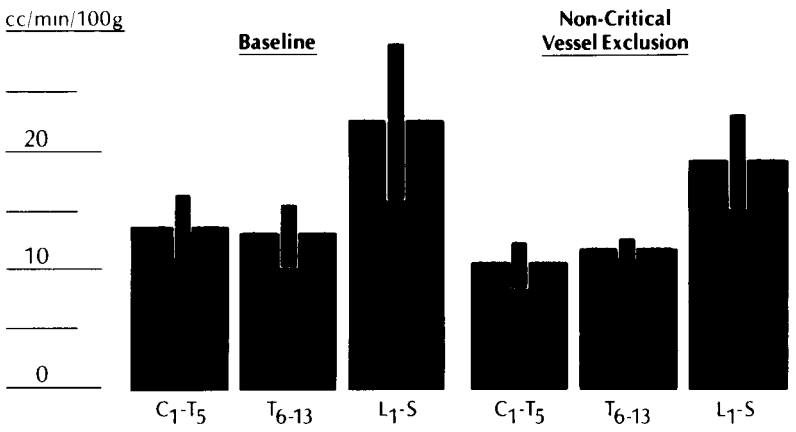

Figure 6. Summary of spinal cord blood flow studies in Group I animals. No changes in spinal cord blood flow were seen due to the maintenance of critical vessel perfusion.

Table 3. Effect of Critical Vessel Exclusion on Somatosensory Evoked Potentials in Group II Dogs (n = 7)

	Latency (ms)	Amplitude (μV)
Baseline	35.24 ± 2.83	4.23 ± 0.68
Proximal exclusion	32.00 ± 3.12	4 10 ± 0.66
Critical vessel exclusion	—	—

Table 4. Use of Somatosensory Evoked Potential Monitoring for Identification of Vessels Critical to Spinal Cord Blood Supply in Group II Dogs

Dog	Aortic Segment Excluded*	Ischemic Spinal Cord Segments†
1	L_6–L_7	T_6–T_{13}
2	T_7–T_{10}	T_6–T_{13}
3	L_3–L_6	L_1–L_3
4	L_3–L_6	T_6–T_{13}
5	T_{13}–L_3	L_1–L_3
6	T_{13}–L_3	L_1–S
7	T_{13}–L_3	T_6–T_{13}

* Lowest aortic segment excluded at the time of somatosensory evoked potential (SEP) loss; † Identified by spinal cord blood flow changes observed at the time of SEP loss.

Abbreviations as in Table 2.

changes. Although some overlap in the spinal cord blood flow distribution by way of the systemic and bypass circulations was observed in both animals, the proximal spinal cord (above T_{13}) was supplied primarily by vessels originating proximal to the left subclavian artery. Lower spinal cord tissue in these animals (below T_{13}) was supplied primarily by vessels perfused by the left heart bypass pump. No ischemic spinal cord segments were noted in either animal.

Discussion

Paraplegia in thoracoabdominal aortic surgery. Although the use of temporary shunt and bypass techniques maintains distal organ perfusion and relieves proximal hypertension after proximal cross-clamping, the efficacy of

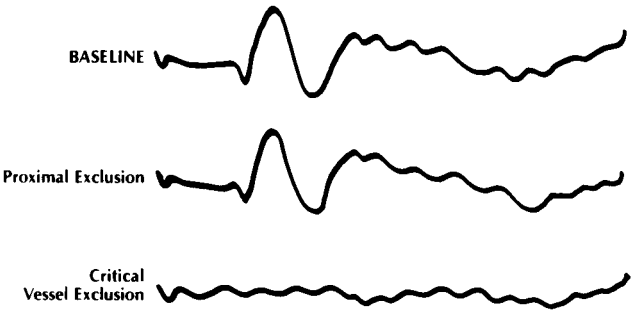

Figure 7. Graphic illustration of somatosensory evoked potential changes in Group II animals. Note that with exclusion of noncritical (proximal) vessels, the trace remains unchanged. Subsequent exclusion of vessels critical to spinal cord blood flow results in loss of somatosensory evoked potentials.

these adjuncts in preventing paraplegia remains uncertain (6–11,15,16). Previous studies from our institution (13,14,17) demonstrated that maintenance of distal aortic perfusion at pressures greater than 60 to 70 mm Hg will preserve spinal cord blood flow after proximal aortic cross-clamping in the absence of critical vessel interruption. The mechanism of paraplegia in patients in whom presumably adequate distal perfusion was provided throughout the cross clamp interval has been attributed to the interruption of vessels critical to spinal cord blood supply (1–3).

Early in the development of thoracoabdominal aortic surgery, it was recognized that inadvertent interruption of vessels critical to spinal cord blood supply could result in paraplegia. In 1958, Spencer and Zimmerman (18) demonstrated that preservation of critical intercostal and lumbar vessels in dogs resulted in prevention of postoperative paraplegia despite the interruption of more proximal blood supply. Subsequent studies in monkeys (19) verified these early experimental results. Clinically, paraplegia has been observed in patients in whom blood flow to vessels critical to the blood supply of the spinal cord has been interrupted during the evolution of acute aortic dissection and in two patients as a complication of intraaortic balloon pumping (20–29 and personal communication). Subsequent clinical experience by Crawford (1), Connolly (11) and their coworkers demonstrated that reimplantation of intercostal or lumbar vessels during the course of operative procedures

Table 5. Effect of Critical Vessel Exclusion on Spinal Cord Blood Flow (cc/100G per min) in Group II Dogs (n = 7)

Spinal Cord Segments	Baseline	Proximal (noncritical) Vessel Exclusion	Critical Vessel Exclusion (somatosensory evoked potential loss)
Above ischemic segment	9 18 ± 0 81	9 19 ± 1 61	12 48 ± 3.49
Ischemic segment	13 84 ± 3 20	14.16 ± 4 17	3.02 ± 0.91*
Below ischemic segment	12 76 ± 2 96	25.76 ± 11 03	19 48 ± 9.03

* $p < 0.0125$ versus baseline.

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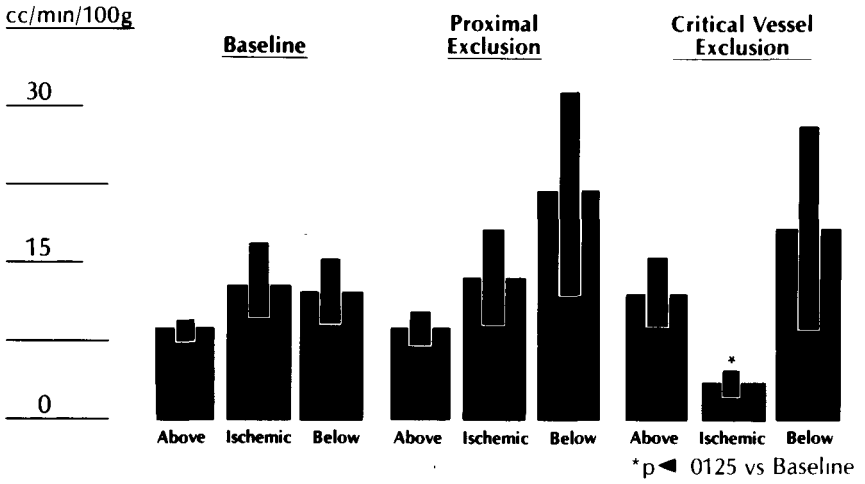


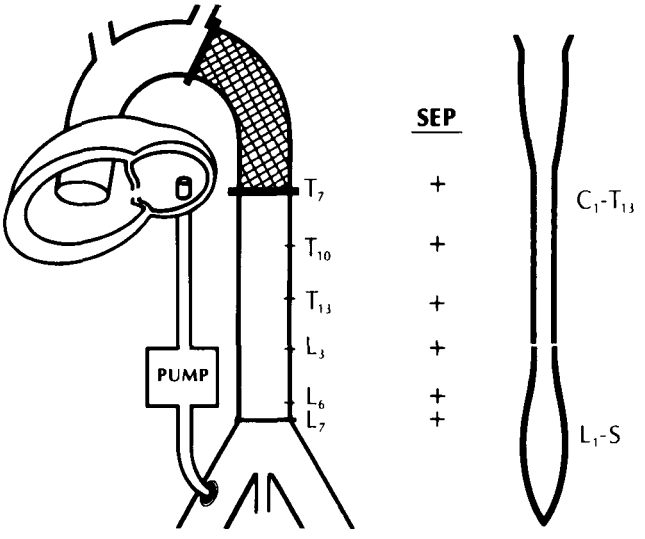
Figure 8. Summary of results of spinal cord blood flow studies in Group II animals. Note that exclusion of proximal vessels noncritical to spinal cord blood flow has no effect on spinal cord perfusion. Subsequent exclusion of vessels critical to spinal cord blood supply results in severe segmental spinal cord ischemia.

on the thoracoabdominal aorta results in reduction of postoperative incidence of paraplegia.

Detection of spinal cord ischemia due to interruption of critical supply vessels. The potential for inadvertent and permanent interruption of blood vessels critical to spinal cord blood supply exists during all procedures on lesions of the descending thoracic or thoracoabdominal aorta (30). It is important to emphasize that the use of adjuncts (that is, temporary shunts or bypass techniques) offers no advantage in preventing paraplegia secondary to interruption of these vessels. The likelihood of critical vessel interruption occurring during any surgical procedure on the descending thoracic aorta stems from the extreme variability in spinal cord blood supply. The vessels critical to spinal cord blood supply can originate anywhere on the descending thoracic and thoracoabdominal aorta, although they are most commonly found between T_8 to L_4 (30,31). Furthermore, the number of critical vessels can vary from one to five. Preoperative angiographic studies only rarely identify vessels supplying important radicals to the anterior spinal artery (32). Efforts to identify and reimplant these arteries intraoperatively have, therefore, been hindered by the failure to reliably identify them both preoperatively and at the time of surgery. This study demonstrates that the technique of intraoperative somatosensory evoked potential monitoring allows accurate and reliable detection of spinal cord ischemia due to the interruption of blood flow to these critical spinal cord vessels. As a result, for the first time, critical vessel interruption is a recognizable event allowing appropriate intervention before the onset of permanent neurologic injury.

Monitoring of somatosensory evoked potentials during aortic surgery. The results of the present study also indicate that intraoperative somatosensory evoked potential monitoring can be used to determine intraoperatively the safety of ligating vessels within the excluded aortic segments. In order to use this monitoring technique for determination of the safety of intercostal vessel ligation and the localization of critical spinal cord vessels, some form of temporary shunt or bypass is necessary. Without these adjuncts, distal spinal cord ischemia routinely occurs, resulting in loss of the evoked potential trace (13,14). Use of these adjuncts assures maintenance of distal aortic and spinal cord blood flow through vessels originating distal to the excluded aortic segment. If somatosensory evoked potentials disappear after exclusion of the diseased aortic segment, the surgeon is alerted that vessels critical to spinal cord blood

Figure 9. Summary of spinal cord blood flow results in Group II animals in whom no somatosensory evoked potential changes were seen despite exclusion of the entire descending thoracic aorta (left subclavian artery - L_7).



flow originate within the excluded segment and that reimplantation of these vessels is necessary to prevent paraplegia. The maintenance of somatosensory evoked potentials after exclusion of large aortic segments confirms the safety of permanent ligation of vessels within the excluded segment without risk of permanent ischemic spinal cord injury. It is also important to note that preservation of evoked potentials accurately predicted the maintenance of normal spinal cord blood flow in two animals despite exclusion of the entire descending thoracic and thoracoabdominal aorta.

Surgical implications. The use of partial bypass techniques after proximal aortic cross-clamping will preserve spinal cord blood flow and function in the absence of critical vessel exclusion. However, even though partial bypass maintains distal aortic perfusion pressure, the exclusion or permanent interruption, or both, of vessels critical to spinal cord blood supply will result in spinal cord ischemia. Intraoperative monitoring of somatosensory evoked potentials allows detection of spinal cord ischemia due to the exclusion of such critical intercostal and lumbar vessels. In addition, the maintenance of such potentials after the exclusion of large aortic segments confirms intraoperatively the development of collateral circulation adequate to maintain normal spinal cord blood flow. Finally, intraoperative monitoring of somatosensory evoked potentials is a highly accurate predictor of the safe lower limits of aortic resection during surgical procedures on the descending thoracic and thoracoabdominal aorta. Clinical trials using this modality for intraoperative localization of vessels critical to spinal cord blood flow are now indicated.

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